STUDY OF CORONARY RISK FACTORS AND CLINICAL PROFILE IN PATIENTS OF ACUTE MYOCARDIAL INFARCTION

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GOPAL GUPTA

C E R T I F I C A T E

This is to certify that the work entitled "STUDY OF CORONARY RISK FACTORS AND CLINICAL PROFILE IN PATIENTS OF ACUTE MYOCARDIAL INFARCTION", which is being submitted as thesis for M.D. (Medicine) examination, 1989 of Bundelkhand University by DR. GOPAL GUPTA, has been carried out in the department of Medicine, M.L.B. Medical College, Jhansi.

He has put in the necessary stay in the department of Medicine as per university regulations.

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Dated: Aug., 1989

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(GOPAL GUPTA)

C O N T E N T S

INTRODUCTION

I N T R O D U C T I O N

Coronary artery disease has assumed epidemic proportions in industrialised western world. In this part of the world about one third deaths in men between 45 to 64 years occur on account of coronary artery disease (CAD). In USA alone about a million people sustain myocardial infarction and about 6 lac people die of CAD every year. Out of these more than half die suddenly. Economic burden of this scourge has been estimated at a staggering figure of about 57 billion US dollar every year.

As against common belief incidence of CAD is not too low in developing world including India. From whatever limited statistics are available in our country, its incidence has been reported from 1 to 6 per thousand population (Barry, 1976). There is every reason to believe that incidence of CAD is fast increasing in India as indeed in other developing countries, possibly due to the increasing prevalence of coronary risk factors as a result of changing life styles (Krishnaswamy, 1970; Dadu Ram, 1984). Clinical studies have shown that ischaemic heart disease constitutes 10-20 of all cardiac admissions in Indian hospitals (Banerjee, 1964). It usually involves individuals in middle and older age groups, however, recently it has been reported that the incidence in younger individuals is also increasing (Gregory, 1983; Gupta et al, 1987).

Diagnosis of atherosclerosis is almost tantamount to the diagnosis of CAD though there are some rare causes also. There are still many unanswered questions regarding aetiopathogenesis of CAD, however, experimental, epidemiological and clinical evidences suggest its multifactorial aetiology. These aetiological factors are termed as "coronary risk factors(CRF)" coronary risk factors vary tremendously from person to person and from society to society dictating incidence of coronary artery disease in that society. In India not many studies have been carried out, especially prospectively regarding various coronary risk factors. In Indian studies the CRF were not found different from western studies but there could still be some unidentified risk factors peculiar to our masses like Bidi smoking and tobacco chewing.

Clinical picture varies widely from patient to patient. On one hand the patient may have very slight chest discomfort and can sustain massive fatal infarction. On the other hand chest pain may be excruciating but there can be only angina or a small infarction. The other features of clinical picture like site, duration, nature of pain etc. are also very variable.

There is wide spread ignorance, apathy among masses regarding the nature, outcome and management of "Heart attack". It is astonishing that for hours or even

days patient resorts to house hold remedies for such a potentially dangerous condition.

Keeping all these facts in mind the present study was carried out in cases of acute transmural myocardial infarction to study :

1. Prevalence of coronary risk factors.
2. Clinical profile of acute myocardial infarction.
3. To assess patient's impression and attitude towards illness.
4. To determine prognostic factors, if any.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Atherosclerosis is the commonest cause of coronary artery disease, being responsible for more than 90%percent cases (Blumgart et al, 1960). A minority of cases (8 percent) are because of coronary artery embolism, inflammatory diseases of coronary arteries like syphilis and congenital anomalous coronary artery lesions (Moritz et al, 1946).

Although any artery may be affected, the aorta, the coronary and the cerebral arteries are the prime targets of atherosclerosis.

In 1958 a study group of WHO defined atherosclerosis as a "Variable combination of changes of the intima of the arteries consisting of focal accumulation of lipids, complex carbohydrates, blood and blood products, fibrous tissue and calcium deposits and associated medial changes" (Cheng, 1974).

Although no agreement has been reached on the precise progression of various changes from the earliest recognisable lesions to the onset of clinically identifiable disease, the following histological abnormalities have been accepted as being present at one time or another in the development of atherosclerotic process (WHO technical Report series No. 143).

1. Patchy accumulation of lipid, mostly cholesterol and its esters but also phospholipids and triglycerides, either intracellularly (foam cells) or extracellularly

in the intima and inner media of affected arteries.

2. Fibroplasia, largely confined to subendothelial portion of the intima, in the form of mucopolysaccharides, reticulin, collagen fibres and hyalinization.
3. Fibrin-like fila attached to the intimal surface or covered by endothelium.
4. Accumulation of complex carbohydrates.
5. Calcification in fine or coarse granules.
6. Cholesterol crystals and fine, granular, amorphous glycoprotein material.
7. Medial changes such as lipid infiltration, disintegration of smooth muscle fibres, disruption of elastic fibres, cellular infiltration, around vasavasorum and mucoprotein accumulation.
8. Secondary changes such as ulceration, thrombosis, or haemorrhage.

AETIOPATHOGENESIS

Ischaemia refers to lack of oxygenation due to inadequate perfusion. Ischaemic heart disease (IHD) is a condition of diverse aetiologies. One factor is common in all conditions i.e. disturbance of cardiac function due to imbalance between oxygen supply and demand.

Ischaemic heart disease was defined by a WHO group in 1955 as "Cardiac disability acute or chronic arising from reduction or arrest of blood supply to the myocardium in association with disease process in the coronary arterial system", (WHO Tech. Report, 1977).

The most common cause of ischaemia is atherosclerotic disease of epicardial coronary arteries, which by reducing lumen of these vessels cause absolute decrease in cross sectional area of a epicardial vessel by approximately 75%percent, a maximal increase in flow to meet increased myocardial demand being not possible. When luminal area is reduced by more than 80 percent, blood flow at rest may be reduced and minor further reduction of stenotic orifice can cause dramatic limitations of coronary flow and severe myocardial ischaemia.

There is disagreement amongst cardiac pathologists concerning the frequency and significance of coronary arterial thrombosis in patients of acute myocardial infarction. Baroldi (1965) found arteriosclerotic narrowing of coronary arteries without recent thrombosis in 53 percent of cases. In patients who died of acute coronary episode suddenly or within 6 hours of initiation of symptoms almost all of these patients had extramural CAHD, but only 10 percent had coronary thrombosis. As a result of these observations Roberts concluded that fresh coronary thrombosis was usually the result and not the cause of myocardial infarction (Roberts, 1972).

Myocardial infarction has been reported in young adults whose coronary arteriogram showed no abnormality (Khan et al, 1974). A possibility is occulusion of artery at its origin which might be missed by arteriography (Glancy, 1971).

Besides atherosclerosis, coronary blood flow can also be limited by arterial emboli (Wenger, 1959) syphilis and inflammation (Moritz, 1946). Cheitlin et al (1975) listed 44 different diseases that may cause myocardial infarction. Myocardial infarction in infants and children is more often related to coronary artery embolism, arteritis or congenitally anomalous vessels than to degenerative disease (Bor, 1969).

PATHOLOGY

Within the first 12-18 hours after the onset of coronary occulusion which leads to infarction, gross changes in the myocardium may not be visible. Recognizable histologic changes are delayed for 5-6 hours and then myocardial fibres appear eosinophilic with fainter cross striations. With new techniques histologic changes may become recognizable within a few hours (Lie, 1978). Fatty infiltration may take place and after 24 hours polymorphonuclear infiltration begins (Maller et al, 1956). After 24-48 hours myocardium grossly appears pale or yellow possibly with haemorrhagic areas. Fibrosis begins after 3rd or 4th week and a healed scar is present as a rule by 6 weeks reaching maximum density by the end of 2nd month.

CORONARY ATHEROSCLEROTIC HEART DISEASE (CAHD)

The concept of "Risk factor" first appeared in an early Framingham study report (Kannel et al, 1961). A risk factor for CAHD is a characteristic of a person

(demographic, psychologic, anatomic or physiologic) that increases the likelihood (risk) of that person developing some manifestations of cardiovascular disease (Kannel, 1984). The risk factor is not only statistically associated with cardiovascular disease, but as a result of meeting several criteria, it is also considered to be causally related to the disease (Susser, 1973).

Risk factors can be divided into 3 groups.

A. Non modifiable risk factors.

B. Modifiable risk factors.

C. Probable risk factors.

NON MODIFIABLE RISK FACTORS

Non modifiable risk factors are age, sex and family history of premature CAHD. These are powerful predictors of HCAHD, but these are not alterable. It may be more important to intervene on modifiable risk factors in males or those with a strong family history.

MODIFIABLE RISK FACTORS

A. Major

1. Elevated serum lipid levels (cholesterol and Triglycerides).
2. Habitual diet high in total calories, total fats, saturated fats, cholesterol, refined carbohydrates and salt.
3. Systemic Hypertension.
4. Smoking.

5. Carbohydrate intolerance.
6. Obesity.

B. Minor

1. Oral contraceptives.
2. Sedentary living habits.
3. Personality type.
4. Psychosocial tension.

C. Possible factors influencing the development of coronary atherosclerosis or its complications.

1. Coffee intake.
2. Alcoholintake.
3. Sucrose intake.
4. Water softness.
5. Urban birth place, residence.
6. Social over crowding.
7. Heavy body frame.
8. Income and living standard.
9. Blood group A.
10. Decreased physical fitness.
11. Hypoxia, carbon monoxide.
12. Carboxy haemoglobin.
13. Alpha radioactivity in water.
14. Decreased stool roughage.
15. Deficiency - vitamin C or E, calcium, magnesium, chromium, magnese, vanadin, lithium or fluoride.

16. Relative or absolute deficiency of copper.
17. Lack of pectin in diet.
18. Abnormal methionine metabolism.
19. Milk antibodies.
20. Immune reaction.
21. Virus infection.
22. Short stature.
23. Respiratory impairment.
24. Decreased vital capacity.
25. Tachycardia at rest.
26. Abnormal ECG at rest or during exercise.
27. Abnormal cold pressure test.
28. Coagulation disorders.
29. Sticky platelets.
30. Elevated haematocrit.
31. Elevated erythrocyte sedimentation rate.
32. Leukocyte count.
33. Axillary hair index.
34. Increased ear canal hair.
35. High levels of circulating insulin.
36. Hyperuricemia.
37. Hypothyroidism (latent).
38. Hypercholesterolemia.
39. Carbon disulphide exposure.
40. Level of education.
41. Birth order.
42. Age of father at birth.
43. Climate.
44. Residence at low altitude.

NON MODIFIABLE RISK FACTORS

AGE

Age has a dominant influence on the development of clinically significant atherosclerosis. Clinically overt atherosclerosis, as evidenced by death rates from ischaemic heart disease, rises with each decade upto age 85 years. So there is a close relationship between age and severity of atherosclerosis (Mc Gill, 1978 and strong, 1978). Other factors such as mode of life, hyponutrition or concomitant wasting diseases, however, can significantly retard the atherogenic process or minimize its invasiveness. This argues in favour of the concept that a relation to age although frequent, is not necessarily involved (Moriyama, 1971). Myocardial infarction is a disease predominantly of the middle aged and the elederly, between 50 and 60 years. The western patients are nearer or over 60 while in India population Kinnare (1982) and Bhusnurmath et al, (1985) found most of the patients between 40-70 years with an average age of 53 years, 30 percent of the patients belonging to age group 50-60 years.

SEX

It is universally accepted that men are more prone to clinical manifestations of coronary atherosclerosis than women of child bearing age. After menopause, there is rapid narrowing of the sex difference in the incidence of

angina pectoris or myocardial infarction and this approaches to equality at about 75 years (Kay, 1970). Of the many reasons presented for sex difference in susceptibility to atherosclerosis, a possible protective effect of oestrogen, differences in blood lipids and haematocrit, reduced risk of cigarette smoking (Dantesen, 1973) and more sheltered way of life are proposed. However, there is no conclusive evidence for any of these. Agrawal et al(1978) observed myocardial infarction in 6.5 percent female and 93.5 percent male patients and Nasir et al (1985) in 10 percent female and 90 percent male patients thus showing dominance of the disease in males.

FAMILY HISTORY OF PREMATURE CORONARY ATHEROSCLEROTIC HEART DISEASE

It has been seen that certain groups have a predisposition for premature coronary atherosclerotic heart disease. It has been confirmed that individuals with either parents or siblings affected by the disease prior to age 50 have a greater risk of coronary atherosclerosis at younger age. In certain cases the relative risk may be as high as 5 times (Fredrickson, 1972). This may represent the clustering of many risk factors within families rather than a unique genetic predisposition to atherosclerosis. In particular hyperlipidemia (genetic or diet induced), hypertension and diabetes, all tend to familial. Family history of CAHD was present in 26 percent

of 175 cases (Nasir et al, 1985). Chinnah et al (1979) found positive family history in 26 percent of his 100 cases and Gupta et al (1987) in 31 percent cases.

Geogery et al (1983) observed that family history of CAHD was more significant in younger patients of acute myocardial infarction(⩽40 years of age) than in older patients. In patients below 40 years he found that the family history was positive in about 65 percent cases in comparison to 32 percent patients above 40 years of age.

MAJOR MODIFIABLE RISK FACTORS

HYPERLIPIDEMIA

There is an overwhelming evidence that hyperlipidemia is associated with increased incidence of premature ischaemic heart disease (IHD). All types of hyperlipoproteinaemias including hypertriglyceridemia and hyperlipoproteinaemia have been correlated with severity of atherosclerosis and the incidence of IHD.

The importance of hypercholesterolemia is associated with age. The Framingham study showed that in men and women 35 to 44 years of age, serum cholesterol levels of 265 mg/100 ml or over were associated with a five times higher risk of developing coronary artery disease than were levels below 220 mg/100 ml. This study also showed that cholesterol levels in males below age 40 were closely related to the future development of IHD. This relation was much less pronounced in older individuals. Low density lipoprotein (LDL) was independently

related to the risk of CAHD for both men and women (Kannel, 1976). In contrast to this high density lipoprotein(HDL) was inversely related to the risk(Castelli,1977).

Patients with high VLDL (very low density lipoprotein) who come from families with familial combined hyperlipidemia appear to be at same increased risk as those members of these families with elevated LDL levels. In contrast, patients with comparably elevated VLDL levels who came from families with pure monogenic familial hypertriglyceridemia do not appear to have a increased risk. In addition, high VLDL may increase the risk of premature atherosclerosis when combined with other risk factors for coronary artery disease such as diabetes, hypertension and patients on chronic haemodialysis. Wasir et al (1985) observed hypercholesterolemia in 37 percent of his 165 cases of acute myocardial infarction. In study of Chinnah et al (1979) out of 100 patients below 40 years 23 percent had high cholesterol, while Gupta et al (1967) found in 19 percent of 40 cases.

HYPERTENSION

Hypertension is a risk factor of prime importance and established association with coronary atherosclerosis (Freis, 1969). Higher the blood pressure, greater is the risk of coronary atherosclerotic heart disease(Alexander, 1975; Kolata, et al, 1976). In the Framingham study, the incidence of IHD in men aged 45 to 62 years with blood

pressure exceeding 160/95 was more than five times than in normotensive men (BP 140/90 or less) (Kannel, 1979).

In the US National Co-operative polling project Research group (1978) which generated data for 10 years period from approximately 7500 men, the risk of IHD in individuals with diastolic BP greater han 105 mm Hg was four times than individuals with diastolic BP 84 mm Hg or less.

The fact that predominantly systolic hypertension in the elderly is innocuous is not true. In Framingham study isolated systolic hypertension has been shown to be associated with increased risk of coronary heart disease. There is no indication even in elderly that cardiovascular risk is more closely linked to diastolic than to systolic pressure (Kannel, 1980).

The risk of atherosclerosis appears to be diminished by therapeutic reduction of blood pressure. Recent studies have shown that reduction of diastolic levels that had been greater than 105 mm Hg significantly reduces the incidence of IHD, strokes and congestive heart failure. in men. Even when the with diastolic blood pressures between 90 to 105 mm Hg are similarly maintained on adequate treatment, the incidence of some of these complications may be reduced (Edwin, 1967).

In a study of 100 cases below 40 years of age Chinnah et al (1979) found hypertension in 20 percent cases. In another study of 40 cases by Gupta et al (1967) 15 percent cases were found hypertensive.

DIABETES

Framingham study (Kannel et al, 1979) showed that for all age groups in both sexes, the incidence of cardiovascular diseases is more in diabetics than among nondiabetics. There is at least a two fold increase in the incidence of myocardial infarction in diabetic men as compared to nondiabetic men. For diabetic women the incidence was almost three times than non diabetic women. The approximately two fold increase in the incidence of hypertension among diabetics, particularly in adult females, may increase the risk further. Moreover, it is also frequently associated with obesity and in females with low HDL cholesterol. Both these factors will further enhance the risk. Thus it is difficult to isolate, diabetes mellitus as a single risk factor, since it is well recognised that obesity, hypertension, hyperlipidemia are also frequently present in diabetic patients (Epstein, 1967).

In women the triad of obesity, diabetes and low HDL cholesterol carries an especially high risk for CAHD (Gordon et al, 1977).

Banerjee (1958) found history of diabetes mellitus in 10.3 percent cases in a series of 106 cases while Vytilingham (1964) reported an incidence of 20 percent in 700 cases. Nasir et al (1983) in a study of of 165 young myocardial infarction patients found diabetes in 15 percent cases.

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et al (1971) showed progressive and comparatively synergistic effect of the risk factors (hypertension, cigarette smoking, over weight, elevated cholesterol etc.) In a study of 200 cases, Agrawal et al (1979) found 15 percent patients without any coronary risk factors, 33 percent with single and 52 percent with two or more risk factors. In another study of 165 cases, Nasir et al (1985) observed that 36 percent of young myocardial infarction patients had no modifiable risk factors. This was in sharp contrast to the 18 percent prevalence of absent risk factor in the older myocardial infarction group. In whole of the group, 22 percent patients had no coronary risk factor. Out of all the patients with recognised risk factors, 47 percent had one, 37 percent had two, 12 percent had three and only 4 percent had more than three coronary risk factors.

EPIDEMIOLOGY

The first premortem diagnosis of coronary thrombosis was made by Adam Hammer (1878). Clinical correlation and post mortem features of myocardial infarction were first described by Herrick (1912).

In USA there has been significant decline in CAHD mortality recently. CHAD mortality climbed through the 1950s, plateaued in the 1960s and then declined sharply in 1970s and this trend is continuing (Havlock et al, 1979; Cooper et al, 1978). This trend is for all

DIET

Role of diet in development of IHD remained debatable many years but now most authorities agree that a diet rich in total calories, total and saturated fats, cholesterol, refined sugar and salt is a major coronary risk factor. Although still there are many septics (Mann, 1977) but it is thought that there is a direct relationship between diet, hyperlipidemia and the development of CAHD. This is supported by following facts summarised by Glueck (1978).

1. Dietary cholesterol intake 0-600 mg/day is closely related to plasma cholesterol levels and dietary saturated plasma fatty acids elevate the serum cholesterol levels, whereas polyunsaturated fatty acids reduce them (Hegsteed, 1965 and Rifkin, 1977).

2. Low cholesterol, low saturated fat and high polyunsaturated fat therapeutic diets reproducibly lower plasma cholesterol levels by 10 to 20 percent. (National Diet Heart Study Group, 1968).

3. Populations with sharply lowered dietary cholesterol and saturated fatty acid intake have lower plasma cholesterol levels and reduced IHD incidence (Sinett, et al, 1974).

4. Immigrants from populations having low plasma cholesterol to ones in which it is high, develop cholesterol levels comparable to their host populations. This fact is supported by the study of Japanese emigrants. The gradient for the crucial three variables (Dietary saturated fat, blood lipids and IHD) increased from indigenous Japanese to migrant Japanese to native Caucasians (Key's, 1970).

5. Due to campaigns by various organisations cholesterol intake in the American population has declined since 1970 and the polyunsaturated/saturated ratio in the dietary fat has increased. Concurrently there has been a definite lowering trend in serum cholesterol levels of adult Americans between 1971 to 1974 as compared to levels during 1960 to 1962. (U.S. National Centre for vital and Health Statistics 1977). In the same time period a significant downward trend (20 percent) in IHD mortality occurred among persons 36 to 74 years of age in USA (Wacker, 1977).

SMOKING

Cigarette smoking is one of the most potent risk factors for atherosclerosis. The surgeon General Report (1964) has first established association between cigarette smoking and IHD. In general, the risk of CAHD is - 2-6 times more in smokers than nonsmokers (Aronow, 1973 and Astrup, 1973).

The effect of cigarette smoking is more closely related to the number of cigarettes smoked per day than to the duration of the habit (Kannel, 1981).

It has also been shown that those who quit smoking have only half the risk of myocardial infarction than those who continue to smoke. However, the major influence of smoking is upon incidence of sudden death Those who stop smoking show a prompt decline in risk and may reach the risk level of nonsmokers as early as after the year of obstention (Edwin, 1987). The benefit of quitting cigarette smoking do not extend beyond age 65 years for heart attacks (Gordon et al, 1974).

At autopsy the degree of aortic and coronary atherioscleresis was found to be greater in smokers than non smokers (Strong, 1979).

OBESITY

Obesity has far long been considered as a significant independent coronary risk factor (Stamler, 1967; Hubert, 1983). Obese individuals are more prone to develop hyperlipidemia, systemic hypertension and

diabetes mellitus (Gordon et al, 1977 and 1979). It has also been noted that weight loss is also accompanied by a corresponding reduction in the level of the major atherogenic risk factors (Ashley, 1974). The risk of CAHD in obesity is more obvious because of its association with other risk factors like hypertension, diabetes and hyperlipidemia.

Banerjee (1958) observed that obesity did not have a significant effect on the incidence of IHD. Gregory et al (1983) found obesity in 58 percent of 165 young cases. Wasir et al (1987) found obesity in 15 percent of 300 cases.

MINOR MODIFIABLE RISK FACTORS

ORAL CONTRACEPTIVES

It has been seen that women receiving oral contraceptives have significantly higher risk of CAHD than non users (Deral, 1976). Mann et al (1977) reported 2.8 times increased risk of death from myocardial infarction in women 30-39 years and 4.7 times in women 40 to 44 years, who use oral contraceptives.

SEDENTARY LIVING

Physical activity may be important because it not only reduces risk of CAHD but also improves efficiency of cardiovascular and respiratory system and improves muscle tone. Epidemiological evidence supports an association between physical inactivity and increased

risk of CAHD in men (Paffenbarger, 1977).

In the Framingham study the overall mortality, cardiovascular mortality and CAHD mortality were all inversely related to the level of physical activity (paffenberger, 1977; Kannel et al, 1979). However, this effect was rather modest as compared to other risk factors examined but did persist even when these were taken into account (Kannel, 1970).

PERSONALITY TYPE

The Framingham study showed that Type A women (with competitiveness, impatience, potential for hostility, exaggerated sense of time urgency) developed CAHD in general and angina in particular twice as common as type B women. Type A Framingham working women had the same risk as type A house wives (Haynes, et al, 1980).

Men who exhibited type A behaviour (work overload, suppressed hostility and frequent job change) were found to be at increased risk of CAHD especially in the 55-64 years age range (Morris et al, 1969; Rosenman et al, 1975).

PSYCHOSOCIAL TENSION

Framingham study showed that social and psychosocial tension, anxiety, suppressed hostility are common in women who suffer from CAHD than women of the same age who remained free of CAHD (Haynesa, 1980). Same observations were made by Syme (1975).

EXCESSIVE COFFEE INTAKE

Coffee was once incriminated as a risk factor (Kannel, 1977). However Framingham study showed no association between coffee intake and coronary attacks when cigarette smoking is adequately taken into account (Dember et al, 1974).

MULTIPLE RISK PROFILES

It is acknowledged that CAHD results from a variety of factors, though none has been found to be strictly determinative. The risk associated with any major risk factor varies according to co-existent constellation of other risk factors. For this reason and because a constellation of risk factors provides substantially better risk prediction than any single factor, multivariate risk assessment is recommended (Gordon, 1982).

Large number of prospective epidemiologic studies show that coronary event in an individual with two predisposing risk factors was not simple sum of two individual risk factors but in fact the risk is much higher For example, cigarette smoking is associated with 3-5 fold increase in relative coronary risk and a cholesterol level above 275 mg percent with a 3-5 fold greater risk than a cholesterol level lower than 225 mg percent. When these two risk factors are present in same individual, however, the coronary risk becomes 14 to 16 times (instead of 6-9 times) greater than in an individual free from these risks factors (Brand et al, 1976). Stamler (1967) and Kannel

age groups and for both the sexes. Similar trends are also reported from Australia and Finland.

This decline is attributed to more awareness in public about the health implications of overnutrition, cholesterol values in blood and its consumption, obesity (3.4 percent decline for men and 5.2 percent for women), increased physical activity, effective control of hypertension and declining trend of smoking(Stamler, 1981).

Ischaemic heart disease is believed to be on increase in India as in other developing countries(Modu, 1984), related possibly to the increasing prevalence of coronary risk factors as a result of changing life styles. Clinical studies had estimated that IHD contributes 10-20 percent of all cases of heart diseases in Indian hospitals (Banerjee, 1964). Population based surveys have been rare, the one in Chandigarh (Sarvotham, 1968) showed prevalence rate of 66/1000 males and 63.7/1000 in females in the urban population while low figures of 1 percent have been mentioned in low income suburban population(Barry,1976).

The incidence of myocardial infarction on the other hand was shown to be 1.29/1000 in Rohtak town population (Gupta, 1978) and in 52-62 percent of hospitalised cases of IHD (Banerjee, 1970 and Naik, 1968).

PRODROMAL SYMPTOMS

Many patients with acute myocardial infarction have a history of previous angina pectoris. In a study, Francis et al (1963) found no previous history of angina

pectoris in 52 percent patients with first attack of acute myocardial infarction.

Patients with known CAHD having pattern of stable angina pectoris usually exhibit increased duration and/or frequency of pain or start developing rest angina days or weeks prior to infarction which may be a warning signal of impending attack. One study described prodromal symptoms in 65 of 100 patients. Fifty nine out of these 65 patients had pain as prodromal symptoms. These symptoms began during a period of 2 months to as little as 4 hours before infarction (Solomon et al, 1969).

PRECIPITATING FACTORS

In most patients of myocardial infarction, the onset of infarction cannot be related to unusual effort. However, there is disagreement about whether physical effort can be blamed as a precipitating factor in some instances. Master et al (1941) found that the incidence of acute myocardial infarction following unusual effort was not out of proportion to the percentage of the 24 hour day spend in such effort. Yater and associates(1949) suggested that in some instances physical exertion could have precipitated the attack in the patients with underlying CAHD.

CLINICAL FEATURES

Symptoms of myocardial infarction are quite variable. In the mildest form it may go unrecognised and be disclosed subsequently only by ECG. At the other end

of the spectrum there may be sudden death presumably due to ventricular fibrillation or asystole.

CHEST PAIN

The pain is the most common presenting complaint in patients with myocardial infarction. It may, however, occur without any pain (Roseman, 1954 and Lindberg, 1960). In one study of patients with acute myocardial infarction 10 percent had no pain and 10 percent had very slight or atypical pain (Stokes, 1969). In another study, 25 percent of patients with acute myocardial infarction aged 30-62, did not have any pain (Kannel et al, 1970).

Evans and Suffon (1956) found that atrial fibrillation and hypertension were common in patients with painless infarction. They also reported syncope as the initial symptom in 4 out of 70 patients with painless infarction. Painless infarction is more common in diabetics, elderly and the seriously ill patients (Braunwald, 1967).

Pain of acute myocardial infarction is similar in quality and location to that of angina pectoris but is often more severe and prolonged. Pain may persist from half an hour to a day or so. Pain is seldom of longer duration unless there is some complication e.g. pericarditis or intermittent attacks of recurrent ischaemia.

Pericardial friction rub is found in 6-10 per cent cases of acute myocardial infarction after first few days of infarction, usually on the 2nd or 3rd day(Stevens, 1953). A pericardial friction rub is ordinarily absent

in first 24 hours (Woff, 1962).

Fever often occurs after first 24 hours and the temperature usually does not exceed the normal by 2 to 3°F. Fever usually lasts for few days to a maximum of about a week. In one study fever was found in 150 out of 160 patients with anterior infarction. The maximum morning temperature was 39°C in 99 percent cases (Lofmark et al, 1976).

Derangement of ventricular function in patients with acute myocardial infarction may be manifested by development of diminished and low pitched first heart sound (Adolph et al, 1970), ventricular filling or S3 gallop sound, atrial gallop sound (S4) and paradoxical splitting of 2nd sound (Harvey, 1969; Coh, 1974). There may be prolongation of atypical systolic impulse (late systolic bulge) or an ectopic systolic impulse at peri-apical area during first few days and then may resolve (Heikkila, 1971; Hurst, 1972). The development of apical systolic murmur may be due to papillary muscle dysfunction.

LABORATORY INVESTIGATIONS

Non specific reaction to myocardial injury is associated with polymorphonuclear leukocytosis which appears within a few hours after the onset of chest pain, persists for 3-7 days and often reaches a level of 12000 to 15000 leukocyte/cu.mm. The ESR rises slowly, peaking during the 1st week and some times remains elevated for 1-2 weeks.

There is increased urinary catecholamine excretion which may be induced by acute infarction, pain arrhythmia or heart failure. Plasma hydrocortisone, growth hormone and urinary catecholamines are increased during first few days. There is also impaired glucose tolerance during this period. This impaired glucose tolerance is related to increased catecholamines and growth hormone levels (Lehovitz et al, 1969).

Within an hour after acute myocardial infarction plasma free fatty acid level often increases significantly. Although there is moderate variation in individual patient, the plasma cholesterol level, which reflects predominantly LDL cholesterol tends to decrease slowly for few weeks after acute myocardial infarction, whereas plasma triglyceride level tends to be moderately elevated for a few weeks following a brief decrease (Fredrickson, 1969).

Their levels on first day are close to the levels attained 3 months later (Pyle et al, 1971).

SERUM ENZYMES

Enzymes are released in large quantities into blood from necrotic heart muscle following myocardial infarction. The rate of liberation of specific enzymes after infarction differs from each other. The enzymes which are usually of diagnostic significance are SGOT CPK and LDH. The serum glutamic oxaleacetic acid (SGOT) begins to rise above normal value (3-40 units/l) within 6-12 hours after infarction, reaching a maximum within

1-3 days and remaining elevated usually till 4th and may be upto 8th day. Increase above 40 units is found in more than 97 percent of myocardial infarction (Agress,1960). It is not very specific enzyme because it is also found in skeletal muscle, liver and RBC and damage to these tissues may also liberate this enzyme. Thus in congestive heart failure, shock and hepatitis, this enzyme will be elevated.

CPK is found in heart, brain, and skeletal muscles but not in lungs and liver. Damage to these tissues liberate this enzyme in blood. It can be elevated by strenuous exercise, chronic alcoholism, convulsions, pulmonary disease, cardioversion, cerebrovascular disease and intramuscular injection.

In clinical practice, CPK determination is of little value when the patient with chest pain is receiving intramuscular injections because its level may increase 5-20 times (Meltzer, 1970; Shaft, 1970). The CPK has three isoenzymes namely BB, MB and MM. The CPK-MB isoenzyme has been reported to be both sensitive and specific in myocardial infarction (Rebert, 1973; Wagner, 1973). When more than 2 percent of total CPK is CPK-MB, it is abnormal. Positive responses were not found in cases of CHF, cardiac arrhythmias, unstable angina, pulmonary embolism, and cardioversion. CPK-MB may appear as early as 4-6 hours after the onset of infarction and reaches peak values at 18-24 hours, at the same time as total CPK

activity returns to normal at 48-72 hours (Rapaport, 1977). In myocardial infarction CPK-MB should exceed 3 percent of total CPK, when total CPK exceeds 100 units per litre.

In myocardial infarction, the level of LDH rises during first day, peaks at 3-4 days and returns to normal in 14 days. There are five isoenzymes of LDH, LDH_1 is specific for heart. Its value rises before the rise of total LDH and may rise when there is no rise in total LDH. Increased LDH_1 is a more sensitive indicator of myocardial infarction than total LDH, being raised in more than 95 percent of cases.

Raised serum myoglobin values were found in all 32 cases of acute myocardial infarction studied within 12 hours after onset of chest pain (Reidilin et al, 1978) Serum myoglobin values might reflect the size of myocardial infarction (Kagan, 1975). Myoglobinuria exceeding 5 mg percent was found in all cases of acute myocardial infarction studied. Myoglobinuria often preceded the rise in serum cardiac enzyme levels and was a more sensitive indicator of cardiac muscle necrosis (Bernstein, 1973).

Serum nickel values rose in 72 percent of patients with acute myocardial infarction studied 12-36 hours after its onset. The mechanism of its rise is not known (Sunderman, 1970).

E.C.G.

The ECG is of paramount importance in recognition of myocardial infarction especially when the history is atypical or when the patient is so ill that he is unable to give a proper history (Wilson et al, 1944; Myers, 1949).

However, there are limitations of ECG recognition of myocardial infarction. While the ECG is seldom normal following acute myocardial infarction, the diagnostic changes are present in only 60 percent cases of acute myocardial infarction (Zinn and Conby, 1950). In another study changes were diagnostic in 82 percent but only in 27 percent when there was an already healed infarct (Sullivan et al, 1978).

The earliest changes are hyperacute T wave changes indicating myocardial ischaemia while injury pattern evolution of transmural infarction is an elevation of ST segment in leads facing the infarct area and pathologic Q waves denote necrosis.

In anteroseptal infarction - Q, ST, T wave, changes appear in lead V_1 - V_4.

Anerolateral infarction I, aV_1, V_5, V_6.

Extensive anterior wall I, aVL, V_1-V_6.

Inferior wall MI-II, III, aVf.

True posterior MI - Prominant R wave depressed ST segment and peaked T wave appear in V_1, V_2, V_3.

Rt ventricular infarction is recognised by ST elevation in V4R, V5R, V6R.

Usually for the first few hours or a day or so after chest pain there are only ST and/or T changes and only after then pathological Q waves appear (Wasserburger, 1965) so the serial ECGs are very important for diagnosis of myocardial infarction.

ST segment remains elevated for several days to as long as 1-2 weeks then it settles down in an uncomplicated infarction.

Pathologic Q wave may increase in size for several days or weeks. Later it remains stationary or decrease in size, perhaps due to scarring and decreased size of infarcted area. In an appreciable percent of cases the ECG, returns to normal or nondiagnostic pattern over years following myocardial infarction. Levine (1951) found ECG changes of old infarction in 20 percent cases, Skaeggested (1966) in 34 percent cases and Young (1970) in 35 percent cases only.

Right ventricular infarction is almost never an isolated finding and usually is associated with inferior and/or true posterior infarction and almost never occurs with anterior wall infarction (Robert, 1978).

Typical pain of myocardial infarction is deep and visceral the quality being heaviness, squeezing, acting (Ross et al, 1966). The other presentation can be in the form of tightness, pulling, constriction, burning sensation, bursting discomfort or stabbing. Pain and discomfort are most often present over middle and lower sternum or left precordium. However, it is not uncommon for the distress

to be centered elsewhere. Besides retrosternal and precordial regions, it may be located only in left upper arm, or radiate down the entire left upper extremity, at back over the interscapular region, lower jaw, neck, upper abdomen or both upper limbs. Pain below umbilicus has been described to be noncoronary.

Pain is usually associated with nausea, vomiting, sweating, weakness, giddiness, anxiety, breathlessness and palpitation. Other less common presentation of myocardial infarction with or without pain are sudden onset breathlessness, sudden loss of consciousness, confusional state, sensation of profound weakness, unexplained profuse perspiration the appearance of arrhythmia or merely an unexplained drop in arterial blood pressure. The urge to defecate may be an early symptom of acute myocardial infarction (Schroeder et al, 1976). This may lead to "bed pan death".

PHYSICAL FINDINGS

The majority of patients demonstrate a considerably high blood pressure during pain even though patient is normotensive in past.

It is also seen that most of the patients develop a gradual decline in blood pressure during first few days following myocardial infarction (Kemhilt et al, 1975). In 112 patients with acute myocardial infarction and 96 with cardiac ischaemia, serial BP readings were

made for 72 hours. During 1st hour after hospital admission 31.7 percent had a BP of 160/100 mm Hg or higher and by 6th hour, without specific antihypertensive therapy only 6.3 percent had BP in this range(Gibson,1978).

Most patients seen within 30 minutes of chest pain have an abnormal heart rate and blood pressure (Webb et al, 1972). Sympathetic overactivity as manifested by sinus tachycardia with or without transient hypertension, is more common in patients with anterior infarction. The genesis and significance of sympathetic overactivity in patients with acute myocardial infarction is unknown. In experimental myocardial infarction, sinus tachycardia has been shown to have a possible adverse effect on the ischaemic myocardium and stimulation of sympathetic nerves lowers the ventricular fibrillation threshold (Morris et al, 1972).

Sinus tachycardia in myocardial infarction may also occur due to fever, anxiety, pericarditis, volume depletion, pulmonary embolism and cardioaccelerator drugs. However, persistent sinus tachycardia, is an ominous sign because it is commonly associated with severe left ventricular dysfunction. In some cases it may precede other findings of left ventricular dysfunction (Morris et al, 1972).

Sinus bradycardia with a heart rate of 40-60 beats/min. is sometimes present, especially when there is seen within first hour or two of the even (Rombilt,

1973 and Pantridge, 1974). Various mechanisms have been proposed to explain the early bradyarrhythmias : stimulation of the vagal neuroreceptors in the region of coronary sinus and atrioventricular node, ischaemia of the sinoatrial and atrioventricular nodes and interference with cholinesterase activity by the ischaemic process. Bradyarrhythmias are not only more frequent but also are more serious at the very onset of myocardial infarction, because arterial hypotension is found in the majority of patients with bradycardia and blood pressure is below 80 mm Hg in nearly half of these patients (Pantridge et al, 1974).

The importance of bradycardia in the genesis of serious ventricular tachyarrhythmias, including ventricular fibrillation becomes apparent when on increasing the heart rate which had earlier showed down, ventricular ectopics are abolished (Warren et al, 1976).

MATERIAL AND METHODS

MATERIAL AND METHODS

The study includes 60 patients on cute transmural myocardial infarction admitted in ICCU of M.L.B. Medical College, Jhansi from September, 1987 to May, 1988.

The diagnosis of myocardial infarction was considered only when at least two of the following three criteria were satisfied.

1. Characteristic clinical presentation.
2. Unequivocal ECG changes as suggested by WHO (1959).
3. A rise in serum level of cardiac enzymes SGOT and CPK (Swan et al, 1976).

METHODS

All the patients were subjected to

1. Detailed history.
2. Physical examination.
3. Laboratory investigations.

All the patients were enquired in detail about following risk factors.

1. Age.
2. Sex.
3. Family history of coronary artery disease or sudden death especially in first degree relatives and especially before 55 years of age.

4. Smoking and tobacco chewing.
5. Systemic hypertension.
6. Diabetes mellitus.
7. Hyperlipidemia.
8. Alcohol consumption.
9. Dietary habits regarding intake of fat.
10. salt and sugar.
10. Type and amount of exercise.
11. Oral contraveptives.
12. Psychological (social) tension.

SMOKING

Persons were classified as non smokers only if they had never smoked. Persons who had quit smoking or who currently smoked had their cigarette or bidi consumption semiquantitated by multiplying the number of packs (cigarette 10/pack, bidi 20/pack) of cigarette or bidi smoked/day by the number of years smoked. They were then arbitrarily divided into those above and below 20 pack year.

HYPERTENSION

Hypertension was considered to be present if the patient was on antihypertensive medications on admission or blood pressure was above 160/90 mm Hg at the time of discharge.

SERUM CHOLESTEROL

Serum cholesterol was determined on admission and at the time of discharge following an overnight fast. It was hoped that effect of acute stress of the pain would n not influence these later values. Only patient having serum cholesterol level more than 250 mg% were considered having hypercholesterolemia.

OBESITY

Patients' height and weight were recorded during hospitalisation. Persons who were 20% overweight according to standard tables were considered as obese.

PERSONALITY TYPE

Persons with competitiveness, work overload exaggerated sense of time urgency, impatience and frequent job change were labelled as persons with "Type A" personality.

CLINICAL PROFILE

1. Detailed history regarding time of onset of chest pain, time when first contacted doctor, duration, severity, site, character and radiation of chest pain has been taken.
2. History of prodromal symptoms in preceding days/weeks.
3. History of associated symptoms like nausea, vomiting, ghabrahat, sweating, palpitation, headache, breathlessness, cold extremities, profound weakness, syncope,

belching, choking etc. were taken from all patients.

4. All these patients were examined in detail at the time of admission and daily for subsequent developments during hospital stay, specially for any hypotension, shock, arrhythmias and other complications.
5. A note was made of the time interval between onset of chest pain and hospital admission.
6. Patients were specifically interrogated regarding their impression about their illness.

INVESTIGATIONS

All the patients were subjected to following investigations :

1. CPK (Once-24 hours after onset of the chest pain).
2. SGOT (Once - 24 hours after onset of the chest pain).
3. Blood sugar (Fasting and postprandial - 2 hours after 75 gm glucose.).
4. Serum cholesterol.
5. Serum uric acid.
6. T.L.C., D.L.C., E.S.R. and Haemoglobin.
7. E.C.G. was done on admission, on 1st, 2nd, 3rd , 7th day, at the time of discharge and whenever needed individually.

All the patients were followed up during hospital course for any complications and recurrence of chest pain.

OBSERVATIONS

OBSERVATIONS

The present study was carried out on 60 patients of acute transmural myocardial infarction admitted to ICCU of M.L.B. Medical College, Hospital, Jhansi, U.P. from August, 1987 to May, 1988.

AGE AND SEX

The age and sex distribution is shown in table I. The youngest person was 30 years and the oldest 100 years of age, a saint, with an average age of 53.4 years. The average age of males and females was 53.5 and 52.4 years respectively. There were 55 (91.67%) male and 5(8.33%) female patients in this study.

TABLE I : AGE AND SEX DISTRIBUTION OF CASES.

Age group (years)	Male	Female	Total No. of cases	Percentage
Upto 40	6	-	6	10.00
41 - 50	15	3	18	30.00
51 - 60	17	-	17	28.33
61 - 70	12	2	14	23.33
71 - 80	4	-	4	6.67
80 and above	1	-	1	1.67
Total	55	5	60	100.00

More than 80% of cases were among the age group 40-70 years.

RELIGION

There were 55(91.67%) Hindus and 5(8.33%) muslims.

OCCUPATION

Table II shows the various occupations of the patients. Most patients fell in the categories of businessmen, manual labourer or government servants/ teachers.

TABLE II : OCCUPATION OF THE PATIENTS.

Occupation	No.of cases	Percentage
Service/Teacher	13	21.67
Businessmen	17	28.33
Manual labourer	16	26.67
House wife	4	6.67
Retired	8	13.33
Sadhu	2	3.33
Total	60	100.00

EDUCATION

The details of educational status of patients are given in table III.

Table III : EDUCATIONAL STATUS OF THE PATIENTS.

Education	No.of cases	Percentage
Illiterate	9	15.00
Below Intermediate	40	66.67
Graduate/PG/Teacher/ Professional	11	18.33
Total	60	100.00

FAMILY INCOME

The patients were divided into 4 groups based on the approximate monthly income (Table IV).

TABLE IV : ECONOMIC STATUS OF THE PATIENTS.

Income (in rupees)	No.of cases	Percentage
∠ 1000	13	21.67
1000-2000	14	23.33
2000-3000	13	21.67
7 3000	20	33.33
Total	60	100.00

URBAN/RURAL BACKGROUND

Fourty two (70%) patients belonged to urban areas and 18(30%) belonged to rural areas.

PHYSICAL ACTIVITY

The level of physical activity of patients is shown in table V.

TABLE V : LEVEL OF ACTIVITY OF THE PATIENTS.

Level of Activity	No.of cases	Percentage
House Hold activity	13	21.67
Mild physical activity	19	31.67
Moderate physical activity	20	33.33
Very active	8	13.33
Total	60	100.00

RISK FACTORS

Table VI : CORONARY RISK FACTORS IN CASES OF MYOCARDIAL INFARCTION.

Risk Factors	No.of cases	Percentage
Smoking	44	73.33
Diabetes	8	13.33
Hypertension	5	8.33
Hypercholesterolemia	13	21.67
Family history of IHD/MI/ sudden death	18	30.00
Family history of IHD/MI/ sudden death ≤55 years	10	16.67
Obesity	9	15.00
Type A personality	22	36.67
Serum Uric Acid (≥ 6 mg%) (done in 33 cases)	5	15.00
Family history of diabetes, hypertension, CVA.	17	28.33

The single most important risk factor was smoking seen in 44 (73.33%) cases. Other risk factors are shown in table VI.

SMOKING

TABLE VII : DETAILS OF SMOKING HABIT OF THE CASES.

Smoking habit	No.of cases	Percentage
A. Non smokers	16	26.67
B. Smokers	44	73.33
a. Bidi smokers	34	77.20
i. ≥20 pack year	21	61.76
ii. ≤20 pack year	13	38.24
b. Cigarette smokers	10	22.80
i. ≥10 pack year	9	90.00
ii. ≤10 pack year	1	10.00

There was no specific predilection for any specific brands. Most of smokers kept chaning their brands.

HABIT AND DIETARY CHARACTERISTICS OF THE PATIENTS

Table VIII and IX show the habit and dietary characteristics of the patients.

TABLE VIII : HABIT CHARACTERISTICS OF THE PATIENTS.

		No.of cases	Percentage
1.	Alcohol Intake		
	Teetotallers	50	83.33
	Occasional	3	5.00
	Regular	7	11.67
2.	Tobacco Chewing	15	25.00
3.	Tea Consumption		
	No tea	5	8.33
	⩽ 5 cups/day	40	66.67
	⩾ 5 cups/day	15	25.00

TABLE IX : FOOD HABITS OF THE PATIENTS.

			No.of cases	Percentage
1.	Vegetarian/Nonvegetarian			
	Vegetarian		47	78.33
	Non vegetarian		13	21.67
2.	Water source	: Tap water	46	76.70
		: Well water	14	23.30
3.	High salt intake	: ⩾10 g/day	5	8.33
4.	Sugar intake	: ⩾100 g/day	6	10.00
5.	Predominant Cooking Media			
	Oil :		41	68.34
	: Mustard	- 26		
	: Sun flower	- 7		
	: Ground nut	- 8		
	Margerine(vanaspati		13	21.67
	Desi ghee		1	1.66
	Uncertain		5	8.33

TABLE X : NUMBER OF RISK FACTORS PER PERSON.

No.of risk factors	No.of cases	Percentage	Percentage of those having coronary risk factors (n=56)
Nil	4	6.67	-
1	25	41.67	44.50
2	19	31.67	34.00
3	7	11.66	12.50
7≥	5	8.33	9.00

Most of the patients had one (41.67%) or two (31.67%) risk factors (Table X). Mean risk factor score in our patients was 1.73.

In our study there were 5 (8.33%) female patients. Risk factors present in these females are given below. Out of these 3 patients were having having diabetes, 2 each were hypertension and hyper cholesterolemia. (Table XI).

TABLE XI : FACTORS (RISK) IN FEMALE PATIENTS.

Patient	Age (years)	Risk factors present
1	45	Diabetes
2	46	Diabetes, Hypercholesterolemia
3	48	Diabetes, hypertension.
4	60	Hypertension.
5	62	Hypercholesterolemia.

RISK FACTORS IN YOUNG ADULTS(⩽40 years)

In our study 6(10%) patients were below 40 years. Risk factors in them are given below. It was noted that all the six patients were smokers.

TABLE XII : RISK FACTORS IN YOUNG ADULTS(⩽40 years).

Patient	Age(in years)	Risk factors
1	30	Smoking
		Family history of IHD
2	39	Smoking
3	39	Smoking
		Hypercholesterolemia
4	36	Smoking
5	38	Smoking
6	38	Smoking

PRECIPITATING FACTORS

Precipitating factors were identifiable only in 27(45%) cases. The following factors were associated with initiation of chest pain : physical exertion in 15(25%), heavy meal in 9(15%) and straining during defaecation in 3(5%).

Emotional disturbances did not apparently precipitate myocardial infarction in any case.

PRODROMAL SYMPTOMS

Prodromal symptoms in the form of brief chest pain, belching, dyspnoea, choking, pain over cheek and

teeth were present for varying period of time ranging from few hours to several days before onset of the severe chest pain of myocardial infarction. These were present in 28 (46.66%) cases (Table XIII).

TABLE XIII : PRODROMAL SYMPTOMS.

Symptoms	No.of cases	Percentage
Chest pain	24	40.00
Belching	1	1.66
Dyspnoea	1	1.66
Pain over cheek and teeth	1	1.66
Choking sensation	1	1.66
Total	28	46.64

TIME OF ONSET OF SEVERE CHEST PAIN

Chest pain started at different times of the day in individual patients. About 30% of patients had an attack during early morning hours between 4 AM to 8 AM (Table XIV)

TABLE XIV : TIME OF ONSET OF CHEST PAIN OF MYOCARDIAL INFARCTION.

Time by watch	No.of cases	Percentage
4 - 8 A.M.	17	28.33
8 - 12 Noon	8	13.33
12 - 4 P.M.	11	18.33
4 - 8 P.M.	10	16.67
8 - 12 Night	6	10.00
12 - 4 A.M.	8	13.33

TIME INTERVAL BETWEEN BEGINNING OF CHEST PAIN AND FIRST CONTACT WITH DOCTOR

About 83 percent of patients contacted the doctor within 6 hours of the onset of chest pain, but only 30 percent contacted within an hour. The smallest time interval was 10 minutes seen in one patients and largest 2 days in one patient. Average time interval between onset of chest pain and first contact with doctor was about 5 hours (4 hours, 50 minutes) (Table XV).

TABLE XV : TIME INTERVAL BETWEEN ONSET OF CHEST PAIN AND FIRST CONTACT WITH DOCTOR

Time Interval	No.of cases	Percentage
10 min. - 1 hr	18	30.00
1 hr - 3 hrs	22	36.66
3 hrs - 6 hrs	13	21.66
6 hrs - 24 hrs	5	8.33
24 hrs	2	3.33

DURATION OF CHEST PAIN

Duration of chest pain varied from 15 min. to 24 hours. More than 50 percent of patients had chest pain varying from 3 to 10 hours. Average duration of pain was about 6 hours (5.55 hours).

TABLE XVI: DURATION OF CHEST PAIN.

Duration	No.of cases	Percentage
15 - 30 min.	4	6.66
30 - 60 min.	5	8.33
1 hr - 3 hrs	7	11.66
3 hrs - 5 hrs	12	20.00
5 hrs - 10 hrs	18	30.00
10 hrs - 15 hrs	6	10.00
15 hrs - 24 hrs	7	11.66

INTERVAL BETWEEN BEGINNING OF CHEST PAIN AND HOSPITAL ADMISSION

About 81 percent patients were admitted within 24 hours of chest pain and 41.67% within 6 hours. No patient could attend the hospital within an hour of chest pain. One patient came after 12 days and two more patients came after 3 days. Excluding these three patients there was an average delay of 12 hours in hospitalisation after the onset of chest pain(Table XVII).

TABLE XVII : INTERVAL BETWEEN CHEST PAIN AND HOSPITAL ADMISSION.

Interval	No.of cases	Percentage
Within an hour	-	-
1 - 6 hours	25	41.67
6 - 12 hours	10	16.67
12 - 24 hours	14	23.33
1 day- 3 days	8	13.33
7 3 days	3	5.00

SITE OF CHEST PAIN

All but two patients had pain in front of chest as given below. One had only epigastric pain and another patient who presented with breathlessness had no chest pain at all (Table XVIII).

TABLE XVIII : SITES OF CHEST PAIN.

Sites	No.of cases	Percentage
Only Retrosternal	10	16.67
Retrosternal + Left chest	24	40.00
Left chest	5	8.33
Whole chest (Front)	10	16.67
Upper half of Front of chest	5	8.33
Lower half of front of chest	4	6.67
Only epigastric	1	1.66

The commonest site of pain was retrosternal area plus left chest (24 cases, 40%) (Table XVIII).

RADIATION OF PAIN

TABLE XIX : SITE OF RADIATION OF CHEST PAIN.

Site of radiation	No.of cases	Percentage
Both arms	16	26.67
Upto left shoulder	13	21.67
Left arm (only)	11	18.33
Neck, jaw and teeth	4	6.67
Back	3	5.00

Radiation of pain away from the chest was observed in 36 (60%) cases. The common sites for

radiation of pain were both arms, left shoulder and left arm alone in order of frequency (Table XIX).

CHARACTER OF CHEST PAIN

Most of the patients described pain as heaviness, constriction, stabbing, piercing or burning over chest. Other types are detailed in table XX).

TABLE XX : CHARACTER OF CHEST PAIN.

Character of chest pain	No. of cases	Percentage
Heaviness	17	28.33
Constriction	7	11.67
Stabbing	7	11.67
Piercing	6	10.00
Burning	5	8.33
Bursting	4	6.66
Discomfort	4	6.66
Pulling	3	5.00
Choking	2	3.33
Squeezing	2	3.33
Cutting	1	1.66
Pulsating	1	1.66

EFFECT OF SUBLINGUAL NITRATE

Chest pain of acute myocardial infarction did not disappear in any of 34 patients who were given sublingual sorbitrate. It decreased slightly in 14(23.33%) and there was no effect in 20(33.33%) cases (Table XXI).

TABLE XXI : EFFECT OF SUBLINGUAL NITRATE ON PAIN.

Effect of S/L NITRATE ON PAIN	No.of cases	Percentage
Disappeared	-	-
Decreased	14	23.33
No effect	20	33.33
Not used	26	43.34

ASSOCIATED SYMPTOMS

Associated symptoms were present in all cases, the common ones being sweating, ghabrahat(uneasiness), vomiting, profound weakness, breathlessness, palpitation and sense of impending death.

TABLE XXII : ASSOCIATED SYMPTOMS IN MYOCARDIAL INFARCTION

Symptoms	No.of cases	Percentage.
Sweating	49	81.66
Sense of impending death	42	70.00
Ghabrahat	33	55.00
Cold extremities	25	41.60
Profound weakness	23	38.30
Breathlessness	22	36.66
Vomiting	21	35.00
Palpitation	14	23.33
Headache	13	21.60
Syncope	12	20.00
Nausea	10	16.67
Belching	4	6.67
Choking sensation	1	1.66
Passage of urine and stool	1	1.66

PATIENT'S OWN IMPRESSION ABOUT THE CAUSE OF HIS ILLNESS

We asked all the patients about their own impression regarding the cause of their illness as they interpreted. More than half (56.6%) of the patients could not form a definite opinion about its cause. All patients with a previous history of angina or myocardial infarction were able to recognise that the chest pain was cardiac in origin. The patients who thought the pain to be due to cold were all admitted in winter season. Other different patient's interpretations are given in table XXIII.

TABLE XXIII : PATIENT'S OWN IMPRESSION ABOUT THE CAUSE OF HIS ILLNESS.

Patient's Impression	No. of cases	Percentage
No definite impression	34	56.60
Cardiac (Angina/M.I.)	10	16.67
Due to heavy meal	2	3.33
'Gas' trouble	3	5.00
Effect of excessive cold	6	10.00
Due to overexertion	1	1.67
Hyperacidity	1	1.67
Hypertension	1	1.67
Faulty sitting posture	1	1.67
Excessive talking	1	1.67

PAST HISTORY

Many patients gave past history of angina, myocardial infarction or other illnesses (Table XXIV).

TABLE XXIV : PREVIOUS ILLNESSES OF THE PATIENTS.

Diseases	No.of cases
Myocardial Infarction	5
Angina Pectoris	15
: Of 1-3 months duration - 7	
: Of 7 3 months duration - 8	
Hypertension	5
Diabetes	8
C.V.A. (Hemiplegia)	1
Chronic obstructive airway disease	4
Bronchiectasis	3
Peptic dyspepsia	1

TABLE XXV : THE AREA OF HEART INVOLVED IN MYOCARDIAL INFARCTION.

Area Involved	No.of cases	Percentage
A. Anterior wall	39	65.00
- Anteroseptal - 20		
- Extensive anterior wall - 13		
- Anterolateral - 6		
B. Inferior wall	19	31.66
C. True posterior wall	1	1.67
D. Inferior + Anterolateral	1	1.67

The commonest area involved in the infarction process was anterior wall(65%) followed by the inferior wall (31.66%). There was only one case with true posterior wall infarction. One had inferior with anterolateral wall infarction.

COMPLICATIONS

In 18.33% of cases no complications were observed. Complications observed in the remaining patients are given in table XXVI.

TABLE XXVI : COMPLICATIONS IN THE PATIENTS OF ACUTE MYOCARDIAL INFARCTION.

Type of complications	No.of* cases	Percentage
Arrhythmias	28	46.66
Left Ventricular failure	10	16.66
Congestive heart failure	5	8.33
Cardiogenic shock	5	8.33
Post infarction angina	15	25.00
Pericarditis	3	5.00
Cerebrovascular accident	2	3.33
Psychosis	1	1.66
Intractable Hiccup	1	1.66
No complications	11	18.33

* Some patients had more than one complications.

Five patients died, 2 suddenly presumably due to ventricular fibrillation, 2 due to cerebral embolism and 1 due to cardiogenic shock. All the deaths occurred within first week of infarction but none on first day.

ARRHYTHMIAS/CONDUCTION DISTURBANCES

Arrhythmias/conduction disturbances were observed in 46.6% patients. The details are given in table XXVII.

TABLE XXVII : TYPES OF ARRHYTHMIAS/BLOCKS IN MYOCARDIAL INFARCTION PATIENTS.

Type of Arrhythmia	No.of* cases (%)	Ant. wall M.I.	Inf. wall MI
Sinus tachycardia(>100/min.)	20(33.33)	15	5
Sinus bradycardia(<60/min)	2(3.33)	1	1
Nodal ectopics	1(1.66)	1	-
Atrial ectopics	2(3.33)	1	1
Paroxysmal Atrial tachycardia	1(1.66)	-	1
Ventricular ectopics	10(16.67)	8	2
Accelerated idio-ventricular rhythms	2(3.33)	2	-
AV dissociation	1(1.66)	1	-
First degree trio ventricular block	-	-	-
Second degree Atrio ventricular block.	2(3.33)	1	1
Complete heart block	-	-	-
Right bundle branch block	6(10.00)	4	2
Right bundlebranch block + Left anterior hemiblock	4(6.66)	3	1
Right bundle branch block + Left posterior hemiblock	1(1.66)	1	-
Left anterior hemiblock	4(6.66)	4	-
Left bundle branch block	-	-	-
Left posterior hemiblock	1(1.66)	1	-

* Some patients had more than one arrhythmias.

DISCUSSION

DISCUSSION

AGE AND SEX

Myocardial infarction is a disease predominantly of middle aged and the elderly. We found most of the patients between 40-70 years with an average of 53 years. Similar results were also seen by Kinnare (1982) and Bhuvanmath et al (1985). In our study 10% of patients were below 40 years of age. This is in similarity with 8.2% by Wig and Malhotra (1951) and Vytilingam (1964). However Wasir et al (1985) found relatively high incidence of myocardial infarction below 40 years (21%). Myocardial infarction occur predominantly in male population. We foun that 91.61% of the patients were males and only 8.33% females. Similar sex incidence was also reported by Agarwal et al (1978) and Wasir et al (1985) - 6.5% and 10% respectively. All the females who had myocardial infarction were above 45 years of age and had attained menopause. It is in conformity with the fact that female sex hormones protect the heart from development of ischaemic heart disease(IHD).

RELIGION

In India the percentage of Hindu, Muslim and Christian population is as follows : Hindus - 86.46%, Muslims - 10.63%, Christians - 2.09% (Census of India, 1971). In our study there were 91.67% Hindus and 8.33% Muslims and no Christians. The incidence of myocardial

infarction in Hindus and Muslims was correlated with their population ratio. These results were in contrast to the results of Vakil (1949) who reported higher incidence in minority community. Vytilingam (1964), Chinnah et al (1979) showed higher incidence in Muslims 15.4% and 20% respectively.

The lower number of Muslims in our study can be because of lower Muslim population in Bundelkhand region. Further, their relatively poor socio-economical and educational status might be responsible to lower admission of myocardial infarction cases from that community. This region, especially Jhansi city though has high percentage of christian population but not a single patient in our study was christian. This can be explained by the fact that there is one St. Jude's hospital in Jhansi, run by Christian Missionaries. This being very popular, especially among christian population, patients of acute myocardial infarction might be going there, thus giving a false impression of low incidence of CAD in Christians.

OCCUPATION

In our study 63.33% patients came from the group comprising, service class, businessman, teachers and retired persons. These results are almost similar to those of Banerjee (1958) who reported 77.4% incidence in a similar group of the society.

SOCIO-ECONOMIC STATUS

It is being reported from all over India that ischaemic heart disease is no longer restricted to the rich and well to do but like Ivy is creeping down to lower income groups also (Editorial, JIMA, 1970). We observed 55% incidence in upper and upper middle class people and 45% in lower middle and lower class. This is in contrast to higher incidence (60.7%) in higher socio-economic group, observed by Vytilingham (1964). In another study Chinnah et al (1979) observed that 80% of their patients were from poor socioeconomical status group.

RURAL AND URBAN

We observed that 70% patients belonged to urban and 30% to rural areas. About 80% of Indian population lives in villages. The fact that 70% of patients were from urban background indicates a 13 fold higher incidence of myocardial infarction in urban population. Though higher incidence of myocardial infarction among urban population perhaps due to increased stress of modern urban life is well described in literature but 13 fold higher incidence in our study has to be far from real. Medical college where the study was conducted being situated in urban area, attracts much more urban population. Moreover, because of more illiteracy, superstitions, poverty, misguidance by practicing quacks and poor transport facilities, village folk is tempted

to get treatment at their doorstep rather than coming to medical college.

PHYSICAL ACTIVITY

We observed that 53.33% of our patients were of sedentary habits (house hold activity or mild outdoor activity), 33.33% moderately active and 13.33% were very active. However, Roth et al (1967) and Chinnah et al (1979) reported lower incidence of sedentary habits in cases of acute myocardial infarction.

FOOD HABITS OF PATIENTS

	Chinnah et al (1979) (%)	Present study No. (%)
1. Water source :		
Tap water	84	46(76.70)
Well water	15	14(23.30)
Both types of water	1	-
2. Vegetarian/Nonvegetarian :		
Vegetarian	31	47(78.33%)
Nonvegetarian	69	13(21.67)
3. High salt intake (10 g/day)	-	5(8.33)
4. Sugar intake (100 g/day)	-	6(10.00)
5. Predominated cooking media		
Oil	-	41(68.34)
Margarine(Vanaspati)	-	13(23.33)
Desi Ghee	-	1(1.67)
Uncertain	-	5(8.33)

Table gives the various food habits noted by Chinnah et al (1979) and us. Most of the patients in

both the studies used tap water. More patients in our study were vegetarians (78.33%) as compared to the study of Chinnah et al (1979) (31%). This may merely reflect the predominent vegetarian habits of the locality. We have found higher than normal (710 gm/day) salt intake in 5(8.33%) and higher sugar intake (7100 g/day) in 6(10.00%) cases. These parameters were not studied by Chinnah et al (1979) or other workers. The predominent cooking medium was vegetable oil in 41(68.3%) cases and vanaspati, Ghee (Margarine) in 19(23.33%) cases in our study.

SMOKING

Heavy smoking is being incriminated all over the world to be responsible for increasing incidence of ischaemic heart disease(Keys et al, 1970). Smoking was the commonest risk factor observed in our study. It was present in 73.33% cases. No female patient was smoker and all of six cases below 40 years were smoker. Our result was similar (64.4%) to results shown by Banerjee (1958). Similarly Gregory et al (1983) observed that 75% his patients smoked 20 pack year. Findings of Nasir et al (1985) differ from ours in that incidence of smoking in their patients was much less (25%) similarly Agarwal et al (1978) found smoking in 25% cases.

The lower incidence of smoking in series of Nasir and Agarwal can be explained by the fact that

firstly their studies are retrospective and their criteria of calling the patient, smoker was more strict than ours. In our patients among the smokers, 77.2% were bidi smoker and only 22.8% were cigarette smokers. Out of bidi smokers 61.76% were chronic and heavy smokers (20 pack year) while among cigarette smokers 90% smoked more than 10 pack years, however Chinnah et al (1979), found cigarette and bidi smoking in equal number of cases. The above difference can be explained by different smoking habits in Bundelkhand region. As bidi manufacturing is very prominent business in this area, more people probably smoke bidis as compared to cigarettes.

HYPERCHOLESTEROLEMIA

We observed hypercholesterolemia in 21.67% cases. This was much less than prevalence of 32% to 60% reported by other workers (Krishnaswamy et al, 1970 - 46.42%; Chinnah et al, 1979 - 39%, Wasir et al, 1985 - 37% and Gregory et al, 1983 - 60% in young adults and 31% in older patients). This difference could be explained by different dietary habits of Bundelkhand region and time of estimation of serum cholesterol after acute myocardial infarction.

DIABETES MELLITUS

Diabetes mellitus is well known risk factor for CAHD. We observed diabetes in 13.33% of our cases. Our results are in conformity with those of Banerjee

(1958) - 10.3% and Wasir et al (1985) - 15%. Relatively higher prevalence has been shown by Wytilingham (1974), (20%), Agarwal (1978) (18%) and Gregory et al(1983)(32%).

HYPERTENSION

Hypertension is firmly established as a risk factor for CAHD in several studies (Doyle et al, 1964; Dolder et al, 1975; Kannel et al, 1979). Various studies have shown the prevalence of hypertension from 10.5 to 28%. We, however, found lower prevalence rate of 8.33% (Agarwal et al, 1978 - 10.5%; Jan Sievers, 1964 - 23%; Weldon et al, 1967 - 28%). This may be due to lower prevalence of hypertension in our population.

FAMILY HISTORY

In 30% of our cases a positive family history of CAD was present, out of which in only 16.67% it was below 55 years of age. This is in accordance with the observations of Wytilingham (1964) and Wasir et al (1985) who found a positive family history in 26% and 32% cases respectively. Gregory et al (1983) found positive family history of myocardial infarction in 65% of their young patients.

We observed family history of diabetes, hypertension, CVA in 28.33% cases. This was similar to 26% shown by Chinnah et al (1979) in young adults.

HYPERURICEMIA

We observed hyperuricemia in 15% of our cases (5 in 33 done). However none of these patients had clinical gout. Chinnah et al (1979) observed hyperuricemia in 43% of cases, Wasir et al (1985) in 31% and Gupta et al (1987) in only 3% cases. None of them have mentioned about the prevalence of clinical gout in their cases.

PERSONALITY TYPE

We observed type A personality in 36.67% of our patients. Similarly Chinnah et al (1979) observed irritable temperament in 43% of their patients.

OBESITY

Previous investigators have suggested that obesity per se is not a risk factor for CAHD but because of higher incidence of hypertension diabetes, hypercholesterolemia in obese people they are at a higher risk of developing CAD(Banerjee, 1958; Vytilingham, 1964; Truett, 1967 and Tibbiin, 1975). Obesity was observed in 15% of our cases. Wasir et al (1987) also observed obesity in 15.5% cases. Gregory et al (1983) observed obesity in 32% of patients above 40 and 57% in patients below 40 years of age. Our results show low incidence of obesity as compared to western reports which is explained by low incidence of obesity in our population as large.

Four patients (6.67%) had no modifiable risk factor. However, similar findings (6%) were also reported by Gregory et al (1983). This was in contrast to 22% reported by Wasir et al (1985). In another study of young myocardial infarction patients Chinnah et al(1979) found no risk factor in 15% cases.

Of the 56 patients with recognizable risk factors, 25 (44.5%) had one, 19(34%) had two, 7(12.5%) had three and 5(9%) had more than three risk factors. Wasir et al (1985) made similar observations. In their study 47% patients had one, 37% two, 12% three and 4% had more than three risk factors.

Thus higher number (3 or more) of coronary risk factors had not been linked to the tendency to have acute myocardial infarction.

The mean risk factor score in our study was 1.73. While Wasir et al (1985) found it to be 1.4 in patients above 40 and 1.2 in patients of below 40 years of age.

Though the number of females in our study is too small to find out any statistical significance but the incidence of diabetes definitely seems much higher being 60% in females as against 9% in males. Hypercholesterolemia and hypertension each were present in 40% females while in only 20% and 5.5% males respectively.

The mean risk factor score in females as well as in males less than 40 years of age is only 1.4 and 1.5 respectively which is much lesser than over all risk factor score 1.73. Lower mean coronary risk factor score in patients below 40 as compared to more than observed by Wasir et al, 1985 (1.2 ; 1.4). From above finding it appears that especially in females and males $\angle$ 40 years, risk factor scoring may be inappropriate and smoking in males less than 40 and diabetes, hypertension and hypercholesterolemia in females are more important coronary risk factors. It may be pertinent to give more marks to one risk factor than to others while calculating risk factor score.

Comparison of risk factors in our study with other studies.

Risk factors	Present study No.(%)	Agarwal et al (1978) (%)	Chinnah et al (1979) (%)	Wasir et al (1985) (%)	Gupta et al (1987) (%)
Smoking	44(73.33)	25.50	76	25	53
Diabetes Mellitus	8(13.33)	19.00	10	15	5
Hypertension	5(8.33)	10.50	20	33	15
Hypercholesterolemia	13(21.67)	-	33	37	10
Obesity	9(15.00)	36.00	25	-	7
Hyperuricemia	5(15.00)	-	42	33	3
Family history of CAHD	18(30.00)	-	21	26	30
Family history of CVA hypertension, diabetes	17(28.33)	-	26	-	-
Physical Activity :					
Mild	32(53.33)	67.00	12	-	-
Moderate	20(33.33)	33.00	45	-	-
Very active	8(13.33)	-	43	-	-

PRECIPITATING FACTORS

In 45% of our patients we noticed precipitating factors which probably initiated chest pain - physical exertion (23%), Heavy meal (15%), and defaecation (5%). However, Chinnah et al (1979) observed precipitating factors (emotional upset, moderate to strenuous exertion and sexual intercourse) in only 15% cases in the study of acute myocardial infarction in young adults.

In our study we did not come across any case in who emotional upset/sexual intercourse precipitated chest pain.

PRODROMAL SYMPTOMS

We observed prodromal symptoms in 46.66% of cases (chest pain in 24 (40%), belching, pain over cheek and teeth, choking sensation in 1(1.66%) each. Out of 24 patients with chest pain, 19 had developed chest pain for the first time/or there was increase in duration, frequency and severity within a week before the myocardial infarction. Chinnah et al (1979) observed prodromals symptoms in 26% patients.

TIME OF ONSET OF CHEST PAIN

We observed that 48.33% of patients developed chest pain during day time (8 AM to 8 PM) while 51.67% during night time (8 PM to 8 AM). Chinnah et al (1979) reported that 58% of their patients developed chest pain during day time and 42% during the night. There was some predilection for the attack to occur in early hours of

morning as 28.33% of patients developed chest pain between 4 AM to 8 AM.

DELAY IN FIRST CONTACT WITH DOCTOR

In our study only 30% patients contacted to doctor within one hour of the onset of chest pain and about 86% of patients within first 6 hours while the rest contacted even later. Most of the patients first tried some home remedies to get rid off the pain and only when these were not effective, they contacted the doctor. When the pain was mild they ignored it and sought medical attention only when complications occurred. This speaks of the need of public education about the importance of seeking medical advice at the very onset of chest pain.

DELAY IN HOSPITALISATION

None of the patients came to the hospital within first hour of onset of chest pain. 41.67% patients came within 6 hours, 81.67% within 24 hours and rest of them (18.33%) came even later and as late as 12 days. Chinnah et al (1979) noted that 49% of patients came to hospital within 6 hours of onset of chest pain and in the rest 16% within 12 hours, 9% within 24 hours and rest 26% after 24 hours.

There was an average delay of 5 hours in contacting the doctor after beginning of chest pain and average delay of 12 hours in hospitalisation. The main reason of late first contact with doctor and hospitalisation could be cause of lack of awareness in general public. Only 10(16.67%) patients had definite impression regarding

the cause of their symptoms being coronary. All of these patients had angina/myocardial infarction in past. Rest 50% patients (83.33%) had either no definite impression (56.6%) or thought of their pain being because of all sort of imaginary ailments like due to heavy meal, 'Gas' trouble effect of excessive cold, due to overexertion, hyper-acidity, hypertension, faulty sitting posture and excessive talking.

Another cause of late hospitalisation could be poor transport facilities, poor socio-economical status of patients and general apathy against hospitalisation. Seven (11.6%) patients were misdiagnosed by attending doctors so that further complicates the problem.

On careful review of the literature we could not find any studies evaluating these factors i.e. delay in first contact with doctor, patient's own impression regarding his illness, time of the attack by watch.

CLINICAL PROFILE

In our study 96.67% of the patients presented with typical chest pain and 3.33% with atypical pain. Atypical chest pain was observed in 4% of young adult cases by Chinnah et al (1979).

We observed radiation of pain away from chest in 60%. Common sites of radiation were both arms (26.67%), left shoulder (21.67%), left arm only (10.33%), while uncommon sites were back(3%) and neck,jaw and teeth (6.67%).

Most of the patients described chest pain as heaviness (28.33 %), constriction (11.66%), stabbing (11.66%), piercing (10%), and burning (0.33%) in character. Other less common descriptions were bursting, discomfort over chest, pulling, squeezing, cutting, pulsating and choking.

After careful review of literature we did not find any such detailed study of radiation sites and character of chest pain.

PAST HISTORY

Past history of myocardial infarction was present in 5(0.33%) cases and angina pectoris of more than 1 month duration was present in 15(25%) cases. Our results are not in conformity with that of Agrawal et al (1978) who observed past history of myocardial infarction in 19.5% and angina in 23.5% cases. On the other hand Chinnah et al (1979) found similar result (myocardial infarction- 2% and angina 12% cases) though their study being in young adults.

SITE OF INFARCTION

We observed anterior wall infarction in 63% of our patients, inferior wall in 31.66%. True posterior wall in 1.66% and inferior with anterolateral wall infarction in 1.66%. Chinnah et al observed anterior wall infarction in 61%, inferior wall in 33%, subendocardial in 5% and posterior wall infarction in 1% of their cases. We purposely excluded cases of sub-endo cardial myocardial

infarction from our study.

COMPLICATIONS

Eighty one percent of our patients had some complications. The common complications were sinus,tachycardia (33.33%),conduction disturbances(32%), post infarction angina(25%), left ventricular failure(16.67%), ventricular ectopics(16.67%). Other complications observed included congestive heart failure (8.33%), cardiogenic shock (6.66%), cerebrovascular accident, sinus bradycardia, accelerated idioventricular rhythm and sudden death in 3.33% each. However, Chinnah et al (1979) observed complications in only 40% cases. Ventricular extrasystoles and cardiogenic shock in 10 cases each, left ventricular failure in 7, congestive heart failure in 5, conduction disturbances in 5, ventricular fibrillation in 3, ventricular tachycardia in 2, papillary muscle dysfunction in 2 and pericarditis in 2. Incidence of premature ventricular ectopics in our study as well as that of Chinnah et al (1979) is very low as compared to generally considered incidence as high as 90-95%. the reason for pick up rate by ICCU doctors and nurses.

The mortality from acute myocardial infarction varies from 14 to 33% (Banerjee, 1971; Nigam, 1973; Agrawal, 1978). It is difficult to work out the true mortality because of high rate of medically unattended deaths before hospitalisation (Armstrong, 1972). Obviously earlier the patients are admitted to any ICCU higher will be overall mortality. The low mortality rate in our study was probably because of late arrival to hospital (most deaths occur

RELATIONSHIP OF TYPE OF MYOCARDIAL INFARCTION WITH VARIOUS RISK FACTORS AND COMPLICATIONS TO MORTALITY

Type of myocardial infarction	No.of cases	No.of deaths	%tage of deaths(5)
a. Anterior wall	39	4	80
b. Inferior wall	19	1	20
b. True post. wall	1	-	-
d. Anterolateral + inferior wall	1	-	-
RISK FACTORS			
Smokers	44	2	40
Diabetes mellitus	5	1	20
Hypertension	5	1	20
Hypercholesterolemia	1	1	20
Obesity	9	2	40
Family history of myocardial infarction.	10	2	40
COMPLICATIONS PRESENT AT ADMISSION			
Complications present	25	3	12
No complications	35	2	5.7

This table shows higher death rate in patients with anterior wall (80%) than inferior wall infarction (20%). Patients who had some complications on admission (12%) have higher death rate than patients without any complications on admission (5.7%), but due to small number of cases these results can not be statistically evaluated.

during the first hour).

Five (0.33%) of our patients died in hospital, four of them had anterior wall and one had inferior wall infarction. The mean risk factor score in patients who died (1.8%) was slightly higher than the survivors (1.73%). Western reports (Norris, 1968; 1969) have attributed no importance of risk factors in determining mortality. However, Indian workers observed high mortality among smokers, hypertensives, and diabetics (Banerjee, 1971 and Nigam, 1973).

SUMMARY AND CONCLUSIONS

SUMMARY AND CONCLUSIONS

The present study entitled "Study of coronary risk factors and clinical profile in patients of acute myocardial infarction" was performed on 60 consecutive cases of acute transmural myocardial infarction who were admitted in ICCU/Medical wards of M.L.B. Medical College, Jhansi between August, 1987 to May, 1989. All the patients were subjected to detailed history especially for coronary risk factors and clinical profile. Detailed physical examination was done in every case especially looking for complications. All the patients were subjected to different laboratory investigations like blood, sugar, serum cholesterol, serum glutamic oxaloacetic acid, creatinine phosphokinase and serum uric acid. Electrocardiogram was done in every case on 1st, 2nd, 3rd and 7th day and whenever needed.

1. Average age was 53.4 years (range 30-100 years). Six cases were $\angle$40 years of age. More then 80% of cases were between 40-70 years. Fifty five (91.67%) were males and 5(8.33%) females. Average age of males and females was 53.5 and 52.4 years respectively.

2. There were 55 (91.67%) Hindus and 5(8.33%) Muslims. Thirteen patients (21.67%) were service class and teachers, 17(28.67%) were businessmen and 16(26.67%) were manual workers/labourers.

3. Nine (13%) patients were illiterate, majority of patients (66.67%) had education below intermediate, while 18.33% patients were highly educated.

4. Thirteen patients (21.67%) had an family income of 1000 rupees/month, 23.33% between 1000-2000; 21.67% between 2000-3000 and 33.33% were having more than 3000/- rupees per month.

5. Twenty eight (46.66%) patients were not involved in significant physical activity.

6. Four patients (6.67%) did not have any risk factor, while 41.67% had one, 31.67%, two 11.66%, three and 8.33% had more than three risk factors. Mean risk factor score was 1.73.

7. Single most important risk factor was smoking being present in 73.33% cases. Out of all smokers 77.2% were bidi smokers and 22.8% cigarette smokers. Among bidi smokers, 61.76% had smoked more than 20 pack years and in cigarette smokers more than 90% smoked 7 10 pack years. Among cigarette smokers most of them kept changing their brands. All the 6 patients who were of less than 40 years age, were smokers.

8. Diabetes was present in only 13.33% and hypertension in 8.33%. Out of five females three were diabetics and two were hypertensive. Hypercholesterolemia was present in 21.67%, hyperuricemia in 15%, obesity in 15% and type A personality in 36.67% cases.

9. Family history of CAD/sudden death was present in 30% cases. Out of which 16.67% it was present at premature age of less than 55 years.

10. Incidence of alcohol intake was very low being 11.67% - regular and 5% occasional drinkers. Habit of tobacco chewing was present in one fourth cases.

11. Forty seven (78.33%) were vegetarian and 13 (21.67%) non vegetarian. A high percentage of patients (68.34%) were using mustard oil and 21.67% margarine as principal cooking media.

12. In 45% patients some precipitating factor initiated the symptoms and prodromal symptoms during preceding hours or days before the onset of infarction were present in 46.66% cases.

13. There was some predilection for myocardial infarction to occur during early hours of morning between 4 AM to 8 AM in 28.33% cases.

14. There was an average delay of 4.83 hours in contacting the doctor after beginning of chest pain and an average delay of 12 hours in hospital admission.

15. Duration of chest pain varied from 15 minutes to 24 hours, average being 5.5 hours. All but two patients had pain infront of chest. One had epigastric pain and another did not have pain at all. Commonest site of chest pain was retrosternal + left chest (40%) only retrosternal in 16.67% and only left chest in 8.33% cases. Radiation of pain away from chest was present in 60% cases. Character of chest pain was quite variable like heaviness, constriction, piercing stabbing and burning type.

16. In one third patients there was no effect of sublingual nitrate and in 23.33% there was slight decrease in pain. Only 24 patients tried sublingual nitrate.

17. Perspiration was present in 81.66%, sense of impending death in 70%, ghabrahat in 55%, cold extremities in 41.66%, nausea and vomiting present in more than 50% cases, and 21.66% patients complained of headache.

18. On specifically enquiring the patients about their own impression about the cause of illness, 56.6% cases could not form any definite impression. Only 16.67% who were known cases of CAD in the past could suspect the real cause of symptoms. The rest attributed the pain to many imaginary causes like "Gas" trouble, heavy meal, excessive cold, over exertion, hyperacidity, hypertension, faulty sitting posture and excessive talking.

19. Five patients had myocardial infarction in the past, five were diagnosed cases of angina pectoris and five more though undiagnosed gave history of angina in past.

20. In 18.33% cases the post infarction period was uncomplicated. Either arrhythmias or conduction defect was present in 46.66%, significant post infarction angina in 25%, left ventricular failure in 16.66%, congestive cardiac failure and cardiogenic shock in 8.33% each, post infarction pericarditis in 5% and cerebral embolism in 3.33% cases.

21. Right bundle branch block alone was present in 10%, LAH in 6.66%, RBBB + LAH in 1.66% and LPH in 1.66% patients.

22. Mortality rate was 8.33%. All the deaths occurred within first week of infarction, but none on the first day. Out of all five patients who died, two of them had sudden death, two due to cerebral embolism and one due to cardiogenic shock.

We believe that incidence of myocardial infarction in females is still low in our community.

Smoking appears to be the most important risk factor and more so in young people. Diabetes and systemic hypertension appear to be important risk factors in females. Habit of bidi smoking needs further research on the subject.

There is wide spread ignorance and apathy for treatment among masses regarding myocardial infarction. The clinical picture is very variable. Hospital mortality in our series is low probably because of late admissions.

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